REMARKS

Claims 1-20 are currently pending in the case. Claims 1-8, 12-15 and 17-20 have been withdrawn from consideration in the Paper filed on December 1, 2003. Claims 9-11 and 16 are under consideration, and stand rejected under 35 U.S.C. § 102(b) based on arguments laid out in the Final Office Action mailed on October 4, 2004. Applicant thanks the Examiner for the careful examination of this application and respectfully requests reexamination and reconsideration of the case, as amended. Below, Applicant addresses each of the rejections levied in the Office Action and explains why the rejections are not applicable to the pending claims as amended.

Amendments to the Claims

Claim 9 has been amended to include a limitation regarding the fractalkine. As amended, claim 9 specifies that the monoclonal antibody used in the method binds to CX3CR1 or *human* fractalkine. Support for this amendment can be found, for example, on page 31, lines 20-22, of the application as originally filed. Applicant respectfully submits that no new matter is added through the proposed amendment of claim 9.

Claim Rejections – 35 U.S.C. § 102(b)

Claims 9-11 and 16 stand rejected under 35 U.S.C. § 102(b), on the ground that they are anticipated by U.S. Pat. No. 6,013,257 (hereafter the '257 patent) to Pan *et al.* as evidenced by Hoover *et al.* (J. Biol. Chem. 2000, 275(30): 23187-23193).

In the Final Office Action, the Examiner notes that: "example 7 of the '257 patent (column 34, lines 16-59) exemplifies the use of antibodies raised to the full extra-cellular domain of fractalkine for the inhibition of an animal model of multiple sclerosis (EAE). Furthermore, the '257 patent specifically recites the preferred use of monoclonal antibodies (column 7, lines 1-4 in particular) that do not cross-react with other proteins naturally in the presence of fractalkine (column 6, lines 5-9 in particular). The '257 patent teaches that antibodies to fractalkine are included within the scope of fractalkine antagonists (column 6, lines 11-14 in particular) and therapeutic use of anti-fractalkine antibodies as fractalkine inhibitors (column 7, lines 12-17 in particular) and that inflammation associated with multiple sclerosis can be treated with inhibitors

of fractalkine function (column 34, line 60 through column 35, line 14 in particular). Interaction of fractalkine with CX3CR1 is a function of fractalkine and is therefore encompassed by the teachings of the '257 patent."

Applicant respectfully submits that monoclonal antibodies raised against the extracellular domain of human fractalkine do not all inhibit interaction between fractalkine and CX3CR1. As clearly shown by the data reported in the Declaration under 37 CFR § 1.132 which is submitted along with the present Amendment, antibody binding to the extracellular domain of human fractalkine does not necessarily inhibit the interaction between CX3CR1 and fractalkine. More specifically, among the anti-FKN antibodies tested in the experiments described in the Declaration, 6 clones were found to exhibit binding inhibitory activity, while 9 clones showed no inhibitory activity. The Examiner's statement that the interaction of fractalkine with CX3CR1 is a function of fractalkine and is therefore encompassed by the teachings of the '257 patent is no longer sustainable.

It is axiomatic that a prior art reference must teach every element of a claim in order to anticipate that claim. The '257 patent does not disclose a monoclonal antibody which suppresses the interaction between CX3CR1 and neurotactin (fractalkine). The '257 patent fails to teach every element of claims 9-11 and 16. Therefore, the methods of claims 9-11 and 16 are not anticipated by the '257 patent, and, furthermore, could not be rendered obvious by the '257 patent.

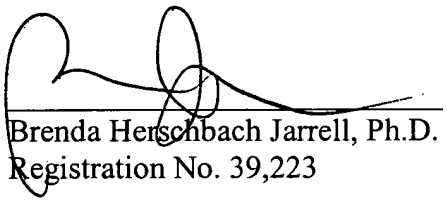
In addition, as discussed on page 13, line 12 to page 14, line 12 of the specification of the application as filed, the present inventors have found that the antibody inhibiting the interaction between CX3CR1 and fractalkine is effective for treating an autoimmune disease. The '257 patent neither discloses nor suggests inhibition of the interaction between CX3CR1 and neurotactin (fractalkine) and advantage thereof. The methods of claims 9-11 and 16 are therefore not anticipated by the '257 patent.

CONCLUSION

Applicant again thanks the Examiner for his careful review of the case. The claims have been amended to obviate all rejections. Based on the Remarks presented above, Applicant respectfully submits that Claims 9-11 and 16 are now in condition for allowance. A Notice to that effect is respectfully requested.

Please charge any fees that may be associated with this matter, or credit any overpayments, to our Deposit Account No.: 03-1721.

Respectfully submitted,



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